



# Medical Nutrition Therapy in CKD

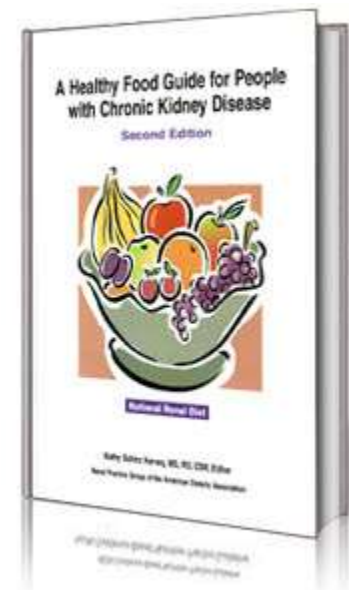
By

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# Outline

- Test your knowledge questions
- CKD & Protein energy wasting
- Main metabolic abnormalities in CKD
- MNT in CKD:
  - Energy
  - Protein
  - Electrolytes



## **1- Causes of PEW in CKD Patients?**

- A- Loss of appetite & diet restrictions
- B- Hyper-metabolism & hormonal disturbances
- C- Co-morbidities & life style
- D- Dialysis factors
- E- All of the above

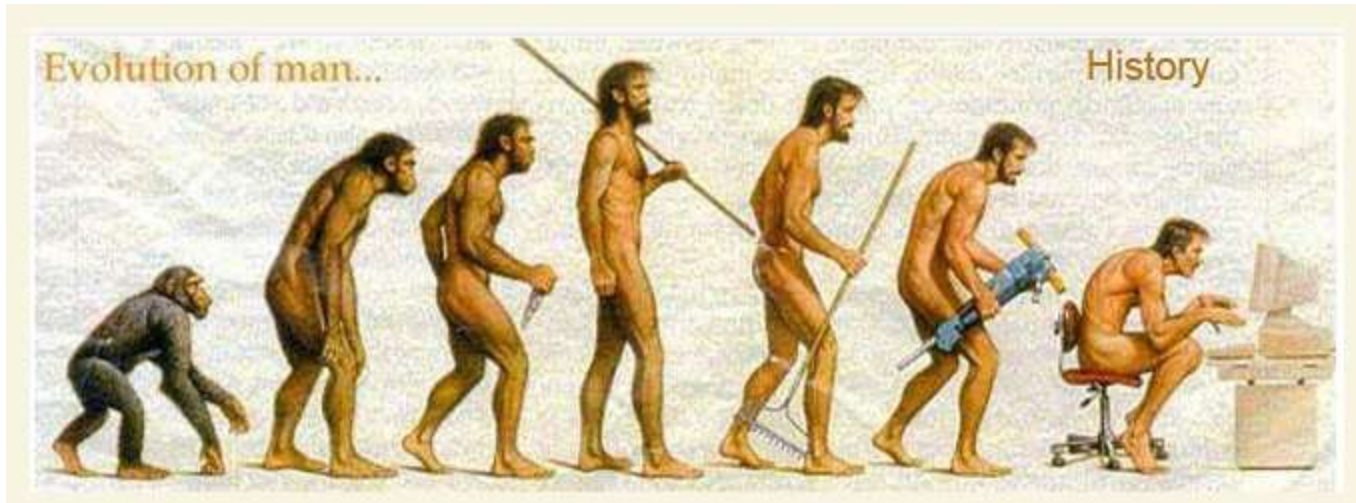
**2- What is the most common cause of death among patients maintained on long-term dialysis?**

A- Uremia

B- Atherosclerotic cardiovascular disease

C- Underlying diseases as DM &HTN

# HISTORICAL DIETARY TREATMENT OF KIDNEY DISEASE



- In the mid-1800s, Richard Bright recommended a milk diet for patients with edema & proteinuria.
- Fishberg in 1930 & Addis in 1948 recommended protein restriction for uremic patients, but neither identified the biological value of the protein
- In 1948, Kempner Rice diet contained about 20 g protein, 150 mg sodium and 2000 calories
- The goal is to preserve life until the kidneys recovered; they were the alternative to dialysis in the 1950s and early 1960s.

- 1960, Rose and Wixon established minimum daily requirements EAA & the importance of balanced meals containing adequate CHO, fat and protein to ensure overall nutrition adequacy.
- In 1963, Giordano applied the concept of high biological value (HBV) protein to the renal diet
- In 1968 and 1973, Kopple 40-g protein diet was more acceptable to the patient because of the greater variety of food selection.

# CKD Definition

CKD is clinically defined as a GFR of  $< 60$  mL/min/1.73m<sup>2</sup> body surface area, with or without evidence of kidney damage, for 3 months or longer.



(Anothaisintawee et al., Clin Neph. 2009)

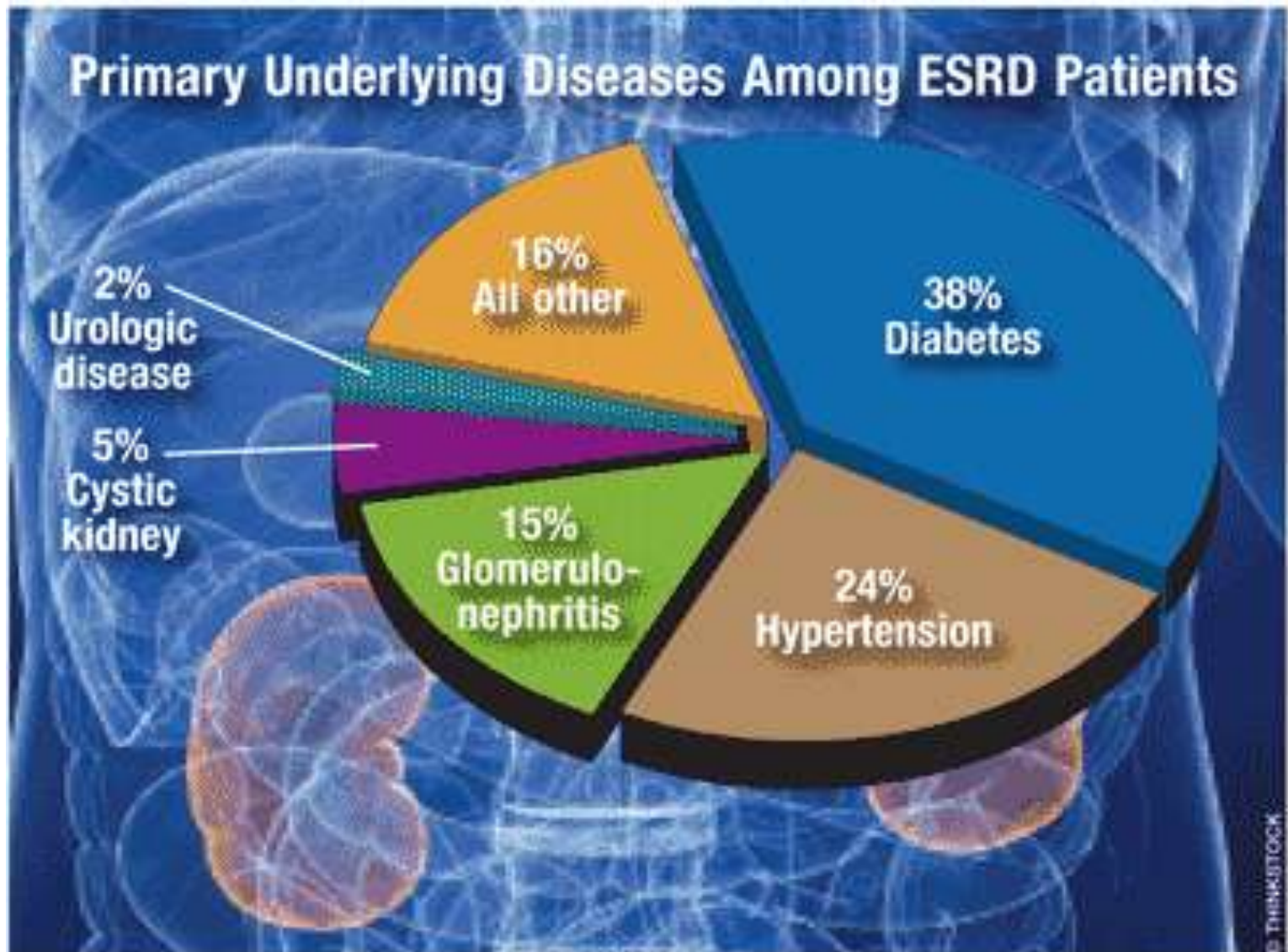


# Stages of CKD

Stage of Disease	Description	GFR (mL/min per 1.73 m <sup>2</sup> )
<b>1</b>	Kidney damage with normal or increased GFR	≥90
<b>2</b>	Kidney damage with mildly decreased GFR	60–89
<b>3</b>	Moderately decreased GFR	30–59
<b>4</b>	Severely decreased GFR	15–29
<b>5</b>	Kidney failure	15 (or undergoing dialysis)

*(Annals of Internal Medicine 2003)*

# Causes of CKD



# PEW in CKD

- **Definition:** Syndrome of adverse changes in nutrition & body composition which is highly prevalent in CKD patients especially in those undergoing dialysis, and it is associated with high morbidity & mortality.

(Carrero et al., J Renal Nutrition: 23;( 2) 2013)

# Causes of PEW in CDK

## 1. Decreased protein and energy intake

a. Anorexia

- i. Dysregulation in circulating appetite mediators
- ii. uremic toxins

b. Dietary restrictions

c. Alterations in organs involved in nutrient intake

d. Depression

e. Inability to obtain or prepare food

## **2. Hyper-metabolism**

- a. Increased energy expenditure
  - i. Inflammation & Increased circulating pro-inflammatory cytokines
  - ii. Insulin resistance secondary to obesity
- b. Hormonal disorders
  - i. Insulin resistance of CKD
  - ii. Increased glucocorticoid activity

## **3. Metabolic acidosis**

## **4. Decreased physical activity**

## **5. Decreased anabolism**

- a. Decreased nutrient intake
- b. Resistance to GH/IGF-1
- c. Testosterone deficiency
- d. Low thyroid hormone levels

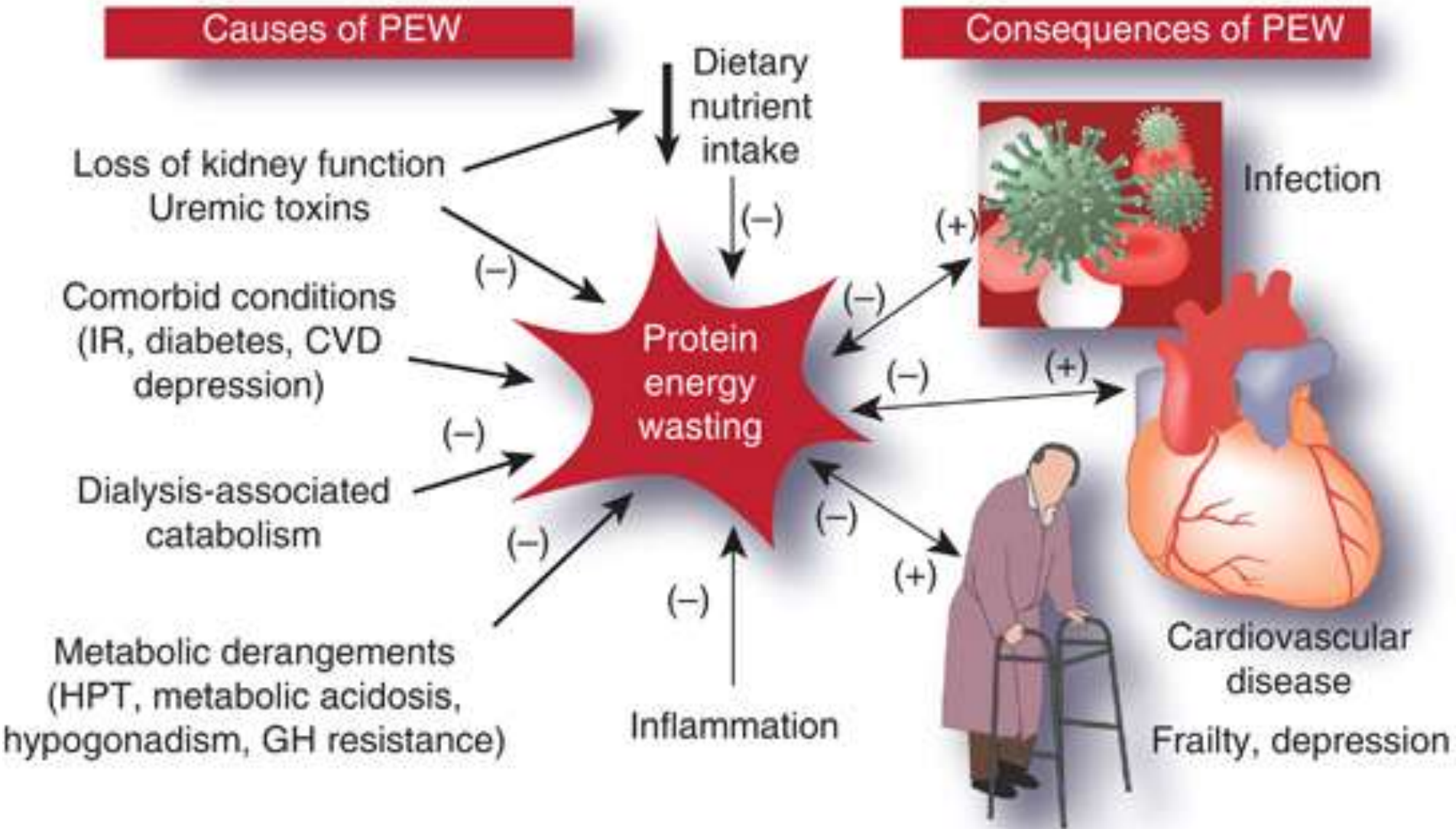
## **6. Co-morbidities & lifestyle**

(DM, CHF, depression, CAD, peripheral VD)

## **7. Dialysis**

- a. Nutrient losses into dialysate
- b. Dialysis-related inflammation
- c. Dialysis-related hypermetabolism
- d. Loss of residual renal function

# Causes & Consequences of PEW



# Assessment & Follow up

- Assessment of nutritional status in CKD requires multiple markers: evaluating protein status, fat stores, body composition, dietary protein & energy intake.
- The nutritional status of patients with CKD should be monitored at regular intervals:
  - Every 1- 3 m for patients with GFR 30 (CKD Stages 4 and 5)
  - Every 6 to 12 m for patients with GFR 30 to 59 (CKD stage 3).



# PEM & Decision of RRT

- The extent of PEM can be considered as an indication for the initiation of kidney replacement therapy.
- If PEM develops or persists despite attempts to optimize protein and energy intake, and there is no apparent cause for malnutrition other than low nutrient intake, initiation of maintenance dialysis or kidney transplant is recommended.

# Diagnosis of PEW in CKD

## Weight Loss:

- Body mass BMI <23 kg/m
- Unintentional weight loss :
  - 5 % over 3 m or 10 % over 6 m
- Total body fat <10 %

## Muscle mass

- Muscle wasting: reduced muscle mass 5 % over 3 m or 10 % over 6 m
- Reduced mid-arm muscle circumference area (>10 % in relation to 50<sup>th</sup> percentile)

## - Dietary intake

- Unintentional low-dietary protein intake  $<0.80$  g/kg/day for at least 2 m for dialysis patients
- or  $<0.6$  g/kg/day for patients on CKD stages 2 to 5
- Unintentional low-dietary energy intake  $<25$  kcal/kg/day for at least 2 m

## Lab:

- Serum albumin < 3.8 g/dl\*
- Serum prealbumin < 30 mg/dL (for maintenance dialysis patients only, levels may vary according to GFR level for patients on CKD stages 2 to 5)
- Serum cholesterol <100 mg/dl\*

# Criteria of Malnutrition in CKD

- International Society of Renal nutrition & Metabolism (ISRNM)
- At least three out of the four listed categories (and at least one test in each of the selected categories) must be satisfied for the diagnosis of kidney disease-related PEW.
- Optimally, each criterion should be documented on at least three occasions, preferably 2-3 weeks apart.

(Fouque D et al. , Kidney Int 2011)

## Lab

- albumin <3.8 g/dL
- Prealbumin <30 mg/dL
- cholesterol <100

## Wt. Loss

- Body mass BMI <23 kg/m
- Wt loss 5 % over 3 m or 10 % over 6 m
- Total body fat <10 % of body wt.

## M Mass

- muscle mass 5 % over 3 m or 10 % over 6 m
- -Reduced mid-arm muscle circumference area >10 %
- Creatinine Appearance

## P or E

- Protein intake <0.6 g/kg/d for at least 2 m in CKD (2-5)
- Energy intake <25 kcal/kg/d for at least 2 m

# Effect of Diet Protein on GFR



- Studies in humans indicate that an increase in GFR can be induced by animal protein and by amino acid mixtures in comparison, vegetable protein and egg whites alone produce little or no effect on GFR.



(Nakamura H et al., Diabetes Care 1993)

# Mechanism of Protein Induced Hyper-filtration

## Main Theories:

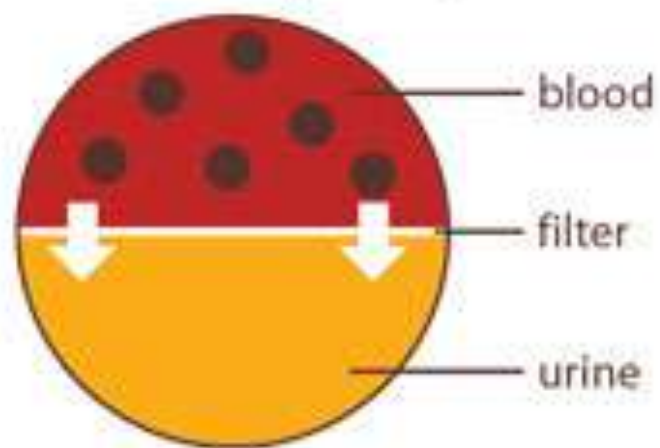
1- Altered release of a hormone or hormones

-  Glucagon (renal vasodilator)
-  IGF-I and kinins

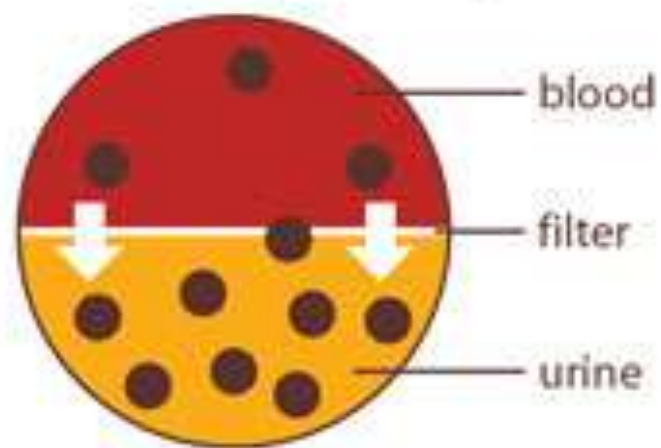
2- Intra-renal Effect:  filtered load of a.a  proximal Na reabsorption via Na-amino acid co-transporters in the proximal tubule.



Inside a *healthy* kidney





Inside a *damaged* kidney



● albumin

# Metabolic Abnormalities in CKD

-  Protein synthesis & breakdown
- Glucose intolerance & diabetes like state
- Abnormal lipid metabolism &  TG
- Electrolyte disturbance
- Altered acid- base & fluid state
- Anemia
- Inflammation & co-morbidities worsen PEW

# Aim of MNT in CKD

- 1- Maintain optimal nutritional status
- 2- Minimize the metabolic complications associating CKD
  - Prevent CVD by treating hyperlipidemia
  - Prevent bone disease
- 3- Retard the progression of CKD



# ESPEN Guidelines

## Daily nutritional requirements in stable CKD patients:

	Conservative therapy	Haemodialysis	Peritoneal dialysis
Energy (kcal/kg/d)	>35	>35	>35
Protein (g/kg/d)	0.6–1.0	1.1–1.4	1.2–1.5
P (mmol)	19–31	25–32	25–32
K (mmol)	38–40	40–63	40–63
Na (mmol)	77–106	77–106	77–106
Fluid (ml)	Not restricted	1000 + DO	1000 ml + DO



# Renal Diet



**In nutrition of patients with CKD there is a delicate balance between induction of toxic effects by giving excess & inducing malnutrition by giving too little.**

# Protein Intake in CKD

- There is insufficient evidence to recommend for or against routine prescription of dietary protein restriction to slow progression
- RDA for protein of 0.75 g/kg/d appears reasonable in patients with GFR 30 mL/min/1.73 m<sup>2</sup> (Stages 1–3).
- 0.6 g/kg/d can be considered for patients with Stages 4 and 5 to slow progression and minimize accumulation of uremic toxins.

- In a renal diet, at least 50% of the protein consumed should be from high-quality.
- Complex CHO, fibers and unsaturated fatty acids.





Individual decision making is recommended after discussion of risks and benefits.

Maintaining adequate energy intake is essential at **all stages of CKD**

# Clinical studies

- MDRD study as well as newly published data showed that LPDs are well tolerated, effective in achieving better metabolic control with fewer drug requirement without evidence of malnutrition
- A small but significant benefit of low-protein intake on renal failure and all-cause mortality after the first 6 years
- However, there was no benefit of protein restriction when outcomes between 6 and 12 years were analyzed.

(HIPPOKRATIA 2011, 15 (Suppl 1))

# Meta-analysis & Systemic review

- A Cochrane Review in 2000 & updated in 2008 (9 randomized controlled trials and 3 before and after studies) evaluated protein-restricted diets in diabetic CKD patients who were followed for at least 4 m.
- These studies provide some evidence that a low protein diet may benefit some patients with CKD
- Limited studies evaluated the effects of a very low protein diet on CKD

“Nutritional studies in patients with CKD suggest that protein intake can be *Safely* lowered to 0.6 g/kg/d  
However, a very low protein diet has been associated with increased mortality over the long term”

(Menon V, Kopple JD, Wang X, et al Am J Kidney Dis 2009)  
(MDRD study)

A low protein diet (0.6 to 0.8 g/kg/d) is recommended in selected predialysis patients who are highly motivated to follow such a diet.

However, the adoption of this diet should **NOT** preclude the initiation of dialysis in patients with severe CKD, if indicated.

# Follow up & Monitoring

- Careful nutritional monitoring of all CKD patients must be maintained if protein-restricted diets are prescribed.
- Three conditions must be met to avoid malnutrition:
  - Adequate caloric intake must be maintained.
  - At least 50 % protein must be of high biologic value
  - Stimulation of skeletal muscle protein breakdown should be prevented to limit net nitrogen loss (metabolic acidosis/resistance Ex.)

Resistance Exercise increase the muscle mass & mitochondrial copy number, increase muscle strength & lowers inflammation





(Castaneda C et al., Ann Intern Med 2001)



# Dyslipidemia

- The key component of dietary intervention is the type and amount of fat consumed with emphasis on reducing saturated and *trans-fatty acid content*.
- Decrease total fat to  $\leq 30\%$  of total calories
- Decrease total cholesterol to  $< 300$  mg/day (i.e., egg yolks & animal fats) substitute whole milk dairy products with skimmed type.



- Decrease saturated fat <7% of total calories
- Encourage use of non-hydrogenated vegetable oils (olive) or nut oils (flaxseed)
- Increasing the consumption of fiber 20–30 g/day   LDL
- MUFA & omega-3 FA   HDL
- Avoid use of *trans fat*



**GOOD**

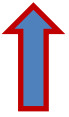
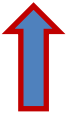







**VS**

**BAD**

**fats**



# Low Phosphate Diet

- “Precipitation-calcification hypothesis” as the main mechanism of renal damage by P.
-  Blood P +  snGFR   Filtered load / renal tubule & trans-epithelial P burden
-  PTH   Ca filtration & reabsorption  
 Precipitation of Ca-P crystals   
Inflammation & Fibrosis

- The daily amount of phosphorus intake was estimated to be at the level of:
  - 16-20 mg/kg/day in usual diet
  - 5-10 mg/kg/day in LPDs
  - 4-9 mg/kg/day in VLPD.
- P rich food mostly of animal origin e.g dairy products, egg yolk & meat

Canned food



Processed food



Instant noodle



High Phosphate

Kailan



Apple



Guava



Low Phosphate

## ***To include more of your favorite vegetables in meals:***

- Use the process of leaching to remove some of the K from vegetables.
- Cut the vegetables into small slices and rinse.
- Soak the vegetables in a large amount of warm water for 2 hours (1:10)
- Cook the vegetables (1:5)



## Spanish



High Potassium

## Onion



Low Potassium



# Counseling





The diet order should be individualized with the least amount of restrictions yet effective to prevent further renal impairment.

# Take Home Message

- PEW is common in CKD patients & if undiagnosed or improperly treated leads to increased morbidity, mortality & lower QOL.
- The causes of PEW are multiple includes:
  - **Decreased protein and energy intake**
  - **Hyper-metabolism**
  - **Metabolic acidosis**
  - **Decreased anabolism & physical activity**
  - **Co-morbidities & lifestyle**
  - **Dialysis**

- Diagnosis of PEW by 3 out of 4 of:
  - Decreased energy & protein intake
  - Decreased of muscle mass
  - Weight loss
  - Lab
- Nutritional assessment & monitoring are essential in all CKD patients

- Modest protein restriction (0.6 to 0.8 g/kg /d) appears to be safe.
- 50 % of protein should be of high biologic value
- Adequate caloric intake must be maintained
- Significant health risks are associated with more severe restrictions over the long term.
- Supervised resistance training, may help maintain muscle mass



# Case

- 64 female patient diagnosed as pneumonia & acute respiratory failure, she has been on HD for 6 years, in the last 5 m, she had several attacks of CVC infection for which she received antibiotics but she lost 8 kg & her weight now 60 kg. She reported inadequate intake in the last 2 m.
- Lab.: Albumin 2.3 (2m ago was 3.5), BUN 118, Cr 3.4, Ca 7.8, K 3.2, P 4.1, TLC 15 with shift to left.
- She is now on daily HD & broad sp antibiotics

- Exam: Loss of temporal & interosseous ms, little SC fat, lax abdomen & good intestinal sounds

Q What is the nutritional diagnosis?

Q Which route of feeding you will choose & why?

Q Which investigations you need while you are following this patient?

A yellow fish is hanging upside down from a metal faucet handle. The fish's mouth is open, and its tail is caught in the handle. The background is a solid blue color.

Knowledge is power but  
Action gets things done

*Reshade.com*